

\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 09:15:44 ON 12 JUN 2001

=> file medline biosis wpids

COST IN U.S. DOLLARS

SINCE FILE  
ENTRY

TOTAL  
SESSION

FULL ESTIMATED COST

0.15

0.15

FILE 'MEDLINE' ENTERED AT 09:16:13 ON 12 JUN 2001

FILE 'BIOSIS' ENTERED AT 09:16:13 ON 12 JUN 2001  
COPYRIGHT (C) 2001 BIOSIS(R)

FILE 'WPIDS' ENTERED AT 09:16:13 ON 12 JUN 2001  
COPYRIGHT (C) 2001 DERWENT INFORMATION LTD

=> s parainfluenza(w)viru?(2)

MISSING OPERATOR 'VIRU?(2'

The search profile that was entered contains terms or  
nested terms that are not separated by a logical operator.

=> s parainfluenza(w)virus(w)2

L1 173 PARAINFLUENZA(W) VIRUS(W) 2

=> s c3396

L2 1 C3396

=> s c3464

L3 1 C3464

=> s c3490

L4 1 C3490

=> s c3457

L5 1 C3457

=> s c3440

L6 1 C3440

=> s l1 and l2

L7 1 L1 AND L2

=> d l7 bib ab

L7 ANSWER 1 OF 1 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD

AN 2000-270817 [23] WPIDS

DNC C2000-082526

TI Attenuated strain of human parainfluenza-2 virus, useful in protective

vaccines, is cold adapted and temperature sensitive so can not grow in the upper respiratory tract.

DC B04 D16

IN BELSHE, R B; NEWMAN, F K

PA (UYSL-N) UNIV ST LOUIS

CYC 88

PI WO 2000012684 A2 20000309 (200023)\* EN 31p

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL  
 OA PT SD SE SL SZ UG ZW

W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES  
 FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS  
 LT LU LV MD MG MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ  
 TM TR TT UA UG US UZ VN YU ZA ZW

AU 9957967 A 20000321 (200031)

BR 9912305 A 20010502 (200129)

ADT WO 2000012684 A2 WO 1999-US19929 19990831; AU 9957967 A AU 1999-57967  
 19990831; BR 9912305 A BR 1999-12305 19990831, WO 1999-US19929 19990831

FDT AU 9957967 A Based on WO 200012684; BR 9912305 A Based on WO 200012684

PRAI US 1998-98667 19980901

AB WO 200012684 A UPAB: 20000516

NOVELTY - an isolated, attenuated strain (A) of human  
**parainfluenza virus-2** (HPIV-2), is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a vaccine containing (A) and a carrier.

ACTIVITY - Antiviral. Seronegative rhesus monkeys were inoculated, intranasally and intratracheally, with the attenuated HPIV-2 strain C3605.

In all cases virus was detected in nasal washes but never in bronchial lavage and no animal had antiviral antibodies in the serum. 56 days later the monkeys were challenged with wild-type HPIV-2. Virus could not be recovered from any of the vaccinated animals and 3 of 4 showed a strong serum antibody response, hemagglutination inhibition titer at least 64 by day 7, while the fourth became seropositive by day 28.

MECHANISM OF ACTION - Vaccine.

USE - (A) is used, particularly in live vaccines, to elicit a protective immune response against HPIV-2 infection (claimed).

ADVANTAGE - (A) are temperature sensitive (unable to grow in the lower respiratory tract) and cold adapted, so stimulate a protective response without the symptoms associated with the wild-type virus.

Dwg.0/2

=> file uspatfull

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	27.02	27.17

FILE 'USPATFULL' ENTERED AT 09:23:55 ON 12 JUN 2001  
 CA INDEXING COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 5 Jun 2001 (20010605/PD)  
 FILE LAST UPDATED: 5 Jun 2001 (20010605/ED)  
 HIGHEST PATENT NUMBER: US8403611  
 CA INDEXING IS CURRENT THROUGH 5 Jun 2001 (20010605/UPCA)  
 ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 5 Jun 2001 (20010605/PD)  
 REVISED CLASS FIELDS (/NCL) LAST RELOADED: Apr 2001  
 USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Apr 2001

>>> Page images are available for patents from 1/1/1997. Current <<<  
 >>> week patent text is typically loaded by Thursday morning and <<<  
 >>> page images are available for display by the end of the day. <<<  
 >>> Image data for the /FA field are available the following week. <<<

```

>>> Complete CA file indexing for chemical patents (or equivalents) <<<
>>> is included in file records. A thesaurus is available for the <<<
>>> USPTO Manual of Classifications in the /NCL, /INCL, and /RPCL <<<
>>> fields. This thesaurus includes catchword terms from the <<<
>>> USPTO/MOC subject headings and subheadings. Thesauri are also <<<
>>> available for the WIPO International Patent Classification <<<
>>> (IPC) Manuals, editions 1-6, in the /IC1, /IC2, /IC3, /IC4, <<<
>>> /IC5, and /IC (/IC6) fields, respectively. The thesauri in <<<
>>> the /IC5 and /IC fields include the corresponding catchword <<<
>>> terms from the IPC subject headings and subheadings. <<<

```

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s 17

```

      895 PARAINFLUENZA
      33286 VIRUS
      2824468 2
        15 PARAINFLUENZA(W) VIRUS(W) 2
        0 C3396
L8      0 L1 AND L2

```

=> s 11

```

      895 PARAINFLUENZA
      33286 VIRUS
      2824468 2
L9      15 PARAINFLUENZA(W) VIRUS(W) 2

```

=> d 19 1-15 bib

```

L9  ANSWER 1 OF 15  USPATFULL
AN  2000:70822  USPATFULL
TI  Interleukin-12 as an adjuvant for paramyxoviridae vaccines
IN  Graham, Barney S., Nashville, TN, United States
    Tang, Yi-Wei, Nashville, TN, United States
PA  Vanderbilt University, Nashville, TN, United States (U.S. corporation)
PI  US 6071893  20000606
AI  US 1997-980160  19971126 (8)
RLI Continuation of Ser. No. US 1994-318480, filed on 5 Oct 1994, now
    abandoned
DT  Utility
EXNAM Primary Examiner: Hauda, Karen M.
LREP Hamilton, Brook, Smith & Reynolds, P.C.
CLMN Number of Claims: 2
ECL  Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 1 Drawing Page(s)
LN.CNT 793
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

```

```

L9  ANSWER 2 OF 15  USPATFULL
AN  2000:7165  USPATFULL
TI  Multiplex PCR assay using unequal primer concentrations to detect HPIV
    1,2,3 and RSV A,B and influenza virus A, B
IN  Henrickson, Kelly J., Oconomowoc, WI, United States
    Fan, Jiang, Wauwatosa, WI, United States
PA  MCW Research Foundation, Milwaukee, WI, United States (U.S.
    corporation)
PI  US 6015664  20000118
AI  US 1996-691045  19960801 (8)
RLI Continuation-in-part of Ser. No. US 1995-552907, filed on 3 Nov 1995,
    now patented, Pat. No. US 5744299, issued on 28 Apr 1998
DT  Utility

```

EXNAM Primary Examiner: Horlick, Kenneth R.; Assistant Examiner: Siew, Jeffrey  
LREP Quarles & Brady  
CLMN Number of Claims: 1  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1846  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 3 OF 15 USPATFULL  
AN 1999:65068 USPATFULL  
TI Porcine parainfluenza virus type 2  
IN Heinen, Ernst, Echternacherbruck, Germany, Federal Republic of  
Schmeer, Norbert, Haan, Germany, Federal Republic of  
Herbst, Werner, Biebertal, Germany, Federal Republic of  
PA Bayer Aktiengesellschaft, Leverkusen, Germany, Federal Republic of  
(non-U.S. corporation)  
PI US 5910310 19990608  
WO 9524214 19950914  
AI US 1996-700548 19960830 (8)  
WO 1995-EP642 19950222  
19960830 PCT 371 date  
19960830 PCT 102(e) date  
PRAI DE 1994-4407489 19940307  
DT Utility  
EXNAM Primary Examiner: Mosher, Mary E.  
LREP Gil, Joseph C.  
CLMN Number of Claims: 6  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1965

L9 ANSWER 4 OF 15 USPATFULL  
AN 1998:45042 USPATFULL  
TI Human parainfluenza virus-1 assay  
IN Henrickson, Kelly J., Oconomowoc, WI, United States  
Fan, Jiang, Wauwatosa, WI, United States  
PA MCW Research Foundation, Milwaukee, WI, United States (U.S.  
corporation)  
PI US 5744299 19980428  
AI US 1995-552907 19951103 (8)  
DT Utility  
EXNAM Primary Examiner: Jones, W. Gary; Assistant Examiner: Rees, Dianne  
LREP Quarles & Brady  
CLMN Number of Claims: 19  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1112  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 5 OF 15 USPATFULL  
AN 97:31566 USPATFULL  
TI Assays for detecting hepatitis B virus envelope antigens or antibodies  
thereto and diagnostic test kits for use in performing the assays  
IN Neurath, Alexander R., New York, NY, United States  
Kent, Stephen B. H., Pasadena, CA, United States  
PA New York Blood, Inc., New York, NY, United States (U.S. corporation).  
California Institute of Technology, Pasadena, CA, United States (U.S.  
corporation)  
PI US 5620844 19970415  
AI US 1993-57200 19930503 (8)  
RLI Division of Ser. No. US 1992-928122, filed on 10 Aug 1992, now  
abandoned  
which is a division of Ser. No. US 1989-337784, filed on 13 Apr 1989,  
now patented, Pat. No. US 5158769 which is a continuation of Ser. No.  
US

1986-856522, filed on 28 Apr 1986, now patented, Pat. No. US 4861588 which is a continuation-in-part of Ser. No. US 485-698499, filed on 5 Feb 1985, now patented, Pat. No. US 4847080 which is a continuation-in-part of Ser. No. US 1984-587090, filed on 7 Mar 1984, now abandoned

DT Utility  
EXNAM Primary Examiner: Caputa, Anthony C.  
LREP Sprung Horn Kramer & Woods  
CLMN Number of Claims: 13  
ECL Exemplary Claim: 1  
DRWN 39 Drawing Figure(s); 23 Drawing Page(s)  
LN.CNT 3170  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 6 OF 15 USPATFULL  
AN 96:94677 USPATFULL  
TI Pre-S gene coded peptide hepatitis B immunogens and synthetic lipid vesicle carriers  
IN Neurath, Alexander R., New York, NY, United States  
Kent, Stephen B. H., Pasadena, CA, United States  
PA New York Blood Center, Inc., New York, NY, United States (U.S. corporation)  
California Institute of Technology, Pasadena, CA, United States (U.S. corporation)  
PI US 5565548 19961015  
AI US 1993-31735 19930315 (8)  
RLI Division of Ser. No. US 1989-338028, filed on 14 Apr 1989, now patented,  
Pat. No. US 5204096 which is a division of Ser. No. US 1985-698499, filed on 5 Feb 1985, now patented, Pat. No. US 4847080 which is a continuation-in-part of Ser. No. US 1984-587090, filed on 7 Mar 1984, now abandoned

DT Utility  
EXNAM Primary Examiner: Warden, Jill; Assistant Examiner: Davenport, A. M.  
LREP Sprung Horn Kramer & Woods  
CLMN Number of Claims: 32  
ECL Exemplary Claim: 1  
DRWN 23 Drawing Figure(s); 14 Drawing Page(s)  
LN.CNT 2561  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 7 OF 15 USPATFULL  
AN 93:31166 USPATFULL  
TI Pre-S gene coded peptide hepatitis B immunogens, vaccines, diagnostics, and synthetic lipid vesicle carriers  
IN Neurath, Alexander R., New York, NY, United States  
Kent, Stephen B. H., Pasadena, CA, United States  
PA New York Blood Center, Inc., New York, NY, United States (U.S. corporation)  
California Institute of Technology, Pasadena, CA, United States (U.S. corporation)  
PI US 5204096 19930420  
AI US 1989-338028 19890414 (7)  
RLI Division of Ser. No. US 1985-698499, filed on 5 Feb 1985, now patented, Pat. No. US 4847080 which is a continuation-in-part of Ser. No. US 1984-587090, filed on 7 Mar 1984, now abandoned

DT Utility  
EXNAM Primary Examiner: Brown, Johnnie R.; Assistant Examiner: Mohamed, Abdel A.  
LREP Sprung Horn Kramer & Wood  
CLMN Number of Claims: 52  
ECL Exemplary Claim: 1  
DRWN 19 Drawing Figure(s); 14 Drawing Page(s)  
LN.CNT 2486  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 8 OF 15 ATFULL  
AN 92:88880 USPTFULL  
TI Pre-S gene coded peptide hepatitis B immunogens, vaccines, diagnostics,  
and synthetic lipid vesicle carriers  
IN Neurath, Alexander R., New York, NY, United States  
Kent, Stephen B. H., Pasadena, CA, United States  
PA New York Blood Center, Inc., New York, NY, United States (U.S.  
corporation)  
California Institute of Technology, Pasadena, CA, United States (U.S.  
corporation)  
PI US 5158769 19921027  
AI US 1989-337784 19890413 (7)  
RLI Continuation of Ser. No. US 1986-856522, filed on 28 Apr 1986, now  
patented, Pat. No. US 4861588 which is a continuation-in-part of Ser.  
No. US 1985-698499, filed on 5 Feb 1985, now patented, Pat. No. US  
4847080 which is a continuation-in-part of Ser. No. US 1984-587090,  
filed on 7 Mar 1984, now abandoned  
DT Utility  
EXNAM Primary Examiner: Brown, Johnnie R.; Assistant Examiner: Mohamed, Abdel  
A.  
LREP Sprung Horn Kramer & Woods  
CLMN Number of Claims: 14  
ECL Exemplary Claim: 1  
DRWN 26 Drawing Figure(s); 16 Drawing Page(s)  
LN.CNT 2992  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 9 OF 15 USPTFULL  
AN 91:64678 USPTFULL  
TI Immunogens containing peptides with an attached hydrophobic tail for  
adsorption to hepatitis B virus surface antigen  
IN Neurath, Alexander R., New York, NY, United States  
PA New York Blood Center, Inc., New York, NY, United States (U.S.  
corporation)  
PI US 5039522 19910813  
AI US 1988-149789 19880129 (7)  
DT Utility  
EXNAM Primary Examiner: Moskowitz, Margaret; Assistant Examiner: Kim, Kay  
LREP Sprung, Horn, Kramer & Woods  
CLMN Number of Claims: 17  
ECL Exemplary Claim: 1  
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)  
LN.CNT 576  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 10 OF 15 USPTFULL  
AN 89:71841 USPTFULL  
TI Pre-S gene coded peptide hepatitis B immunogens, vaccines, diagnostics,  
and synthetic lipid vesicle carriers  
IN Neurath, Alexander R., New York, NY, United States  
Kent, Stephen B. H., Pasadena, CA, United States  
PA New York Blood Center, Inc., New York, NY, United States (U.S.  
corporation)  
California Institute Technology, Pasadena, CA, United States (U.S.  
corporation)  
PI US 4861588 19890829  
AI US 1986-856522 19860428 (6)  
RLI Continuation-in-part of Ser. No. US 1985-698499, filed on 5 Feb 1985  
which is a continuation-in-part of Ser. No. US 1984-587090, filed on 7  
Mar 1984, now abandoned  
DT Utility  
EXNAM Primary Examiner: Moskowitz, Margaret; Assistant Examiner: Mohamed,  
Abdel A.  
LREP Sprung Horn Kramer & Woods

CLMN Number of Claims: 6  
ECL Exemplary Claim: 1  
DRWN 31 Drawing Figure(s); 14 Drawing Page(s)  
LN.CNT 2972  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 11 OF 15 USPATFULL  
AN 89:56230 USPATFULL  
TI Pre-S gene coded peptide hepatitis B immunogens, vaccines, diagnostics,  
and synthetic lipide vesicle carriers  
IN Neurath, Alexander R., New York, NY, United States  
Kent, Stephen B. H., Pasadena, CA, United States  
PA New York Blood Center, Inc., New York, NY, United States (U.S.  
corporation)  
California Institute of Technology, Pasadena, CA, United States (U.S.  
corporation)  
PI US 4847080 19890711  
AI US 1985-698499 19850205 (6)  
RLI Continuation-in-part of Ser. No. US 1984-587090, filed on 7 Mar 1984,  
now abandoned  
DT Utility  
EXNAM Primary Examiner: Moskowitz, Margaret; Assistant Examiner: Mohamed,  
Abdel A.  
LREP Sprung Horn Kramer & Woods  
CLMN Number of Claims: 43  
ECL Exemplary Claim: 1  
DRWN 15 Drawing Figure(s); 14 Drawing Page(s)  
LN.CNT 2496  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 12 OF 15 USPATFULL  
AN 86:43706 USPATFULL  
TI Composition for use in immunoassays  
IN Neurath, A. Robert, New York, NY, United States  
PA New York Blood Center, Inc., New York, NY, United States (U.S.  
corporation)  
PI US 4604348 19860805  
AI US 1984-572494 19840120 (6)  
DCD 20010710  
RLI Division of Ser. No. US 1981-323003, filed on 19 Nov 1981, now  
patented,  
Pat. No. US 4459359, issued on 10 Jul 1984  
DT Utility  
EXNAM Primary Examiner: Wiseman, Thomas G.; Assistant Examiner: Moskowitz, M.  
LREP Sprung Horn Kramer & Woods  
CLMN Number of Claims: 13  
ECL Exemplary Claim: 1  
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)  
LN.CNT 534  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 13 OF 15 USPATFULL  
AN 86:36897 USPATFULL  
TI 2-acetylpyridine thiosemicarbazones as antiviral agents  
IN Shipman, Jr., Charles, Dexter, MI, United States  
Klayman, Daniel L., Chevy Chase, MD, United States  
Smith, Sandra H., Ann Arbor, MI, United States  
Drach, John C., Ann Arbor, MI, United States  
PA The United States of America as represented by the Secretary of the  
Army, Washington, DC, United States (U.S. government)  
PI US 4596798 19860624  
AI US 1982-363723 19820330 (6)  
DT Utility  
EXNAM Primary Examiner: Goldberg, Jerome D.; Assistant Examiner: Rollins,  
Jr.,

John W.  
LREP Gapcynski, William G.; Dautremont, James H.; B. Amy, Werten F. W.  
CLMN Number of Claims: 64  
ECL Exemplary Claim: 1  
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)  
LN.CNT 977  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 14 OF 15 USPATFULL  
AN 85:68150 USPATFULL  
TI Identification and preparation of epitopes on antigens and allergens on the basis of hydrophilicity  
IN Hopp, Thomas P., Seattle, WA, United States  
PA New York Blood Center, Inc., New York, NY, United States (U.S. corporation)  
PI US 4554101 19851119  
AI US 1983-461802 19830128 (6)  
RLI Continuation-in-part of Ser. No. US 1981-223558, filed on 9 Jan 1981  
And  
a continuation-in-part of Ser. No. US 1981-272855, filed on 12 Jun 1981  
And a continuation-in-part of Ser. No. US 1982-358150, filed on 15 Mar 1982  
DT Utility  
EXNAM Primary Examiner: Wiseman, Thomas G.; Assistant Examiner: Teskin, Robin Lyn  
LREP Sprung Horn Kramer & Woods  
CLMN Number of Claims: 6  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1059  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 15 OF 15 USPATFULL  
AN 84:38680 USPATFULL  
TI Sensitive immunoassays of antigens or antibodies sequestered within immune complexes  
IN Neurath, A. Robert, New York, NY, United States  
PA New York Blood Center, Inc., New York, NY, United States (U.S. corporation)  
PI US 4459359 19840710  
AI US 1981-323003 19811119 (6)  
DT Utility  
EXNAM Primary Examiner: Padgett, Benjamin R.; Assistant Examiner: Moskowitz, M.  
LREP Sprung, Horn, Kramer & Woods  
CLMN Number of Claims: 23  
ECL Exemplary Claim: 1  
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)  
LN.CNT 573  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.



> d 15 1-15 ab bib

L5 ANSWER 1 OF 15 MEDLINE

AB Type I interferon (IFN) induces antiviral responses through the activation of the ISGF3 transcription factor complex that contains the subunit proteins STAT1, STAT2, and p48/ISGF3 gamma/IRF9. The ability of some human paramyxoviruses to overcome IFN actions by specific proteolysis of STAT proteins has been examined. Infection of cells with type 2, but not type

1

or type 3 human parainfluenza virus (HPIV) leads to a loss of cellular STAT2 protein. Expression of a single HPIV2 protein derived from the V open reading frame blocks IFN-dependent transcriptional responses in the absence of other viral proteins. The loss of IFN response is due to V-protein-induced proteolytic degradation of STAT2. Expression of HPIV2 V causes the normally stable STAT2 protein to be rapidly degraded, and this proteolytic activity can be partially alleviated by proteasome inhibition.

No V-protein-specific effects on STAT2 mRNA levels were observed. The results indicate that the V protein of HPIV2 is sufficient to recognize and target a specific cellular transcription factor for destruction by cellular machinery. Copyright 2001 Academic Press.

AN 2001326273 MEDLINE

DN 21235793 PubMed ID: 11336548

TI The V protein of **human parainfluenza virus** 2 antagonizes type I interferon responses by destabilizing signal transducer and activator of transcription 2.

AU Parisien J P; Lau J F; Rodriguez J J; Sullivan B M; Moscona A; Parks G D; Lamb R A; Horvath C M

CS Immunobiology Center, Mount Sinai School of Medicine, New York, New York 10029, USA.

SO VIROLOGY, (2001 May 10) 283 (2) 230-9.  
Journal code: XEA; 0110674. ISSN: 0042-6822.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200106

ED Entered STN: 20010611

Last Updated on STN: 20010611

Entered PubMed: 20010504

Entered Medline: 20010607

L5 ANSWER 2 OF 15 MEDLINE

AB A full-length cDNA clone was constructed from the genome of the human parainfluenza type 2 virus (hPIV2). First, Vero cells were infected with recombinant vaccinia virus expressing T7 RNA polymerase, and then the plasmid encoding the antigenome sequence was transfected into Vero cells together with polymerase unit plasmids, NP, P, and L, which were under control of the T7 polymerase promoter. Subsequently, the transfected cells

were cocultured with fresh Vero cells. Rescue of recombinant hPIV2 (rPIV2)

from cDNA clone was demonstrated by finding the introduced genetic tag.

As

an application of reverse genetics, we introduced one nucleotide change (UCU to ACU) to immediate downstream of the RNA-editing site of the V

gene

in the full-length hPIV2 cDNA and were able to obtain infectious viruses [rPIV2V(-)] from the cDNA. The rPIV2V(-) possessed a defective V protein that did not have the unique cysteine-rich domain in its carboxyl terminus (the V-protein-specific domain). The rPIV2V(-) showed no growth in CV-1 and FL cells. Replication of the rPIV2V(-) in these cells, however, was partially recovered by adding anti-interferon (IFN)-beta antibody into the culture medium, showing that the rPIV2V(-) is highly sensitive against IFN and that no growth of rPIV2V(-) in CV-1 and FL cells is mainly due to its hypersensitivity to endogenously produced IFN. These findings indicate that the V-protein-specific domain of hPIV2 is related to IFN resistance. On the other hand, the rPIV2V(-) efficiently replicated in Vero cells, which are known as a IFN-non-producers. However, the virus yields of rPIV2V(-) in Vero cells were 10- to 100-fold lower than those of control rPIV2, although syntheses of the viral-specific proteins and their mRNAs in rPIV2V(-)-infected Vero cells were augmented up to 48 p.i. in comparison with those of rPIV2. Furthermore, the rPIV2V(-) virions showed anomalous in size as compared with rPIV2 virions. These results suggest that the V protein plays an important role in the hPIV2 assembly, maturation, and morphogenesis. Copyright 2001 Academic Press.

AN 2001255000 MEDLINE  
DN 21251393 PubMed ID: 11352671  
TI Recovery of infectious human parainfluenza type 2 virus from cDNA clones and properties of the defective virus without V-specific cysteine-rich domain.  
AU Kawano M; Kaito M; Kozuka Y; Komada H; Noda N; Nanba K; Tsurudome M; Ito M; Nishio M; Ito Y  
CS Department of Microbiology, Mie University School of Medicine, 2-174 Edobashi, Mie, 514-8507, Japan.. kawanom@doc.medic.mie-u.ac.jp  
SO VIROLOGY, (2001 May 25) 284 (1) 99-112.  
Journal code: XEA; 0110674. ISSN: 0042-6822.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 200106  
ED Entered STN: 20010625  
Last Updated on STN: 20010625  
Entered PubMed: 20010515  
Entered Medline: 20010621

L5 ANSWER 3 OF 15 MEDLINE  
AN 2001179666 MEDLINE  
DN 21109096 PubMed ID: 11162793  
TI Inhibition of interferon-mediated antiviral responses by influenza A viruses and other negative-strand RNA viruses.  
AU Garcia-Sastre A  
CS Department of Microbiology, Mount Sinai School of Medicine, New York, New York 10029, USA.. adolfo.garcia-sastre@mssm.edu  
NC AI46954 (NIAID)  
AI48204 (NIAID)  
CA77432 (NCI)  
SO VIROLOGY, (2001 Jan 20) 279 (2) 375-84. Ref: 129  
Journal code: XEA; 0110674. ISSN: 0042-6822.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
General Review; (REVIEW)  
(REVIEW, TUTORIAL)  
LA English  
FS Priority Journals  
EM 200103  
ED Entered STN: 20010404  
Last Updated on STN: 20010404

L5 ANSWER 4 OF 15 MEDLINE

AB Two monoclonal antibodies (mAbs) specific for the human parainfluenza virus type 2 (hPIV-2) V protein were obtained by immunizing mice with the V protein recombinantly expressed in Escherichia coli. Both mAbs were found to react with the V protein in ELISA and in Western blot analysis. Using these mAbs and previously obtained mAbs specific for hPIV-2 nucleoprotein (NP) or hPIV-2 phospho-(P) protein, we examined the intracellular distributions of the V, P and NP proteins in

hPIV-2-infected

cells by indirect immunofluorescence analyses. The P and NP proteins were organized in numerous granules in the cytoplasm of hPIV-2 infected cells. In contrast, the V protein showed diffuse nuclear and cytoplasm distributions.

AN 2000214733 MEDLINE

DN 20214733 PubMed ID: 10753059

TI Isolation of monoclonal antibodies directed against the V protein of human

parainfluenza virus type 2 and localization of the V protein in virus-infected cells.

AU Nishio M; Tsurudome M; Ito M; Kawano M; Kusagawa S; Komada H; Ito Y

CS Department of Microbiology, Mie University School of Medicine, Japan..  
nishio@doc.medic.mie-u.ac.jp

SO MEDICAL MICROBIOLOGY AND IMMUNOLOGY, (1999 Nov) 188 (2) 79-82.  
Journal code: M58; 0314524. ISSN: 0300-8584.

CY GERMANY: Germany, Federal Republic of

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200005

ED Entered STN: 20000512

Last Updated on STN: 20000512

Entered Medline: 20000502

L5 ANSWER 5 OF 15 MEDLINE

AB The simian parainfluenza virus 5 (SV5) V/P gene encodes two proteins: V and the phosphoprotein P. The V and P proteins are amino coterminal for 164 residues, but they have unique carboxyl termini. The unique carboxyl terminus of V contains seven cysteine residues, resembles a zinc finger, and binds two atoms of zinc. In a glutathione-S-transferase (GST)-fusion protein selection of cell lysate assay, the GST-V protein was found to interact with the 127-kDa subunit (DDB1) of the damage-specific DNA binding protein (DDB) [also known as UV-damaged DNA binding protein (UV-DDB), xeroderma pigmentosum group E binding factor (XPE-BF), and the hepatitis B virus X-associated protein 1 (XAP-1)]. A reciprocal GST-DDB1 fusion protein selection assay of SV5-infected cell lysates showed that DDB1 and V interact, and it was found that V and DDB1 could be coimmunoprecipitated from SV5-infected cells or from cells expressing V and DDB1 using the vaccinia virus T7 expression system. The interaction

of

V and DDB1 involves the carboxyl-terminal domain of V in that either deletion of the V carboxyl-terminal domain or substitution of the

cysteine

residues (C189, C193, C205, C207, C210, C214, and C217) in the zinc-binding domain with alanine was able to disrupt binding to DDB1. The V proteins of the mumps virus, **human parainfluenza virus 2 (hPIV2)**, and measles virus have also been found to interact with DDB1 in GST-fusion protein selection assays using in vitro transcribed and translated DDB1.

Copyright 1998 Academic Press.

AN 1998414651 MEDLINE

DN 98414651 PubMed ID: 9740790

TI The V protein of the paramyxovirus SV5 interacts with damage-specific DNA

binding protein.

AU Lin G Y; Paterson R G; Richardson C D; Lamb R A  
 CS Department of Biochemistry, Molecular Biology and Cell Biology, Howard Hughes Medical Institute, Northwestern University, 2153 North Campus Drive, Evanston, Illinois, 60208-3500, USA.

NC AI-23173 (NIAID)  
 T32 GM-08152 (NIGMS)

SO VIROLOGY, (1998 Sep 15) 249 (1) 189-200.  
 Journal code: XEA; 0110674. ISSN: 0042-6822.

CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199810  
 ED Entered STN: 19981021  
 Last Updated on STN: 19981021  
 Entered Medline: 19981013

L5 ANSWER 6 OF 15 MEDLINE  
 AB A recombinant baculovirus expressing the nucleocapsid gene (NP) of Newcastle disease virus (NDV), a member of the genus Rubulavirus, has been generated and shown to express the native protein to high levels in insect cells. In contrast to the NP protein of the rubulavirus **human parainfluenza virus 2**, the NDV protein has been demonstrated by electron microscopy and caesium chloride gradient analysis to be capable of self-assembly in vivo to form nucleocapsid-like structures in the absence of other NDV proteins. These structures, which contained RNA that was resistant to micrococcal nuclease digestion, were also observed when the protein was expressed in E. coli, a phenomenon which was not inhibited by the presence of a 40 amino acid fusion region at the amino terminus of the protein. Further, the formation of these structures was inhibited by the co-expression of the phosphoprotein (P). Therefore, we conclude that the P protein acts as a chaperone, preventing uncontrolled encapsidation of non-viral RNA by NP protein.

AN 97437490 MEDLINE  
 DN 97437490 PubMed ID: 9292023  
 TI Assembly of recombinant Newcastle disease virus nucleocapsid protein into nucleocapsid-like structures is inhibited by the phosphoprotein.

AU Errington W; Emmerson P T  
 CS Department of Biochemistry and Genetics, Medical School, University of Newcastle upon Tyne, UK.

SO JOURNAL OF GENERAL VIROLOGY, (1997 Sep) 78 ( Pt 9) 2335-9.  
 Journal code: I9B; 0077340. ISSN: 0022-1317.

CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199710  
 ED Entered STN: 19971013  
 Last Updated on STN: 19980206  
 Entered Medline: 19971001

L5 ANSWER 7 OF 15 MEDLINE  
 AB Antigenic relationships of simian virus 41 (SV41) to other paramyxoviruses were examined by immunoprecipitation of isotope-labelled SV41-infected cell lysates with specific antisera. SV41 is closely related to the group comprising **human parainfluenza virus 2** (HPIV 2), simian virus 5 (SV5), parainfluenza virus 4 and mumps virus. Slight cross-neutralization was detected between SV41, HPIV-2 and SV5. Anti-SV41 activities were detected in 21 of 1116 human serum specimens, indicating that a proportion of the human population is infected with SV41. The haemagglutinin-neuraminidase of SV41 was

preferentially immunoprecipitated by anti-SV41 positive sera.

AN 91011355 MEDLINE  
 DN 91011355 PubMed ID: 2212992  
 TI Immunological relationships of simian virus 41 (SV41) to other paramyxoviruses and serological evidence of SV41 infection in human populations.  
 AU Nishio M; Tsurudome M; Bando H; Ito Y  
 CS Department of Microbiology, Mie University School of Medicine, Japan.  
 SO JOURNAL OF GENERAL VIROLOGY, (1990 Sep) 71 ( Pt 9) 2093-7.  
 Journal code: I9B; 0077340. ISSN: 0022-1317.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199011  
 ED Entered STN: 19910117  
 Last Updated on STN: 19910117  
 Entered Medline: 19901105

L5 ANSWER 8 OF 15 MEDLINE  
 AB Two strains of **human parainfluenza virus** 2 (HPV2), P2 1972/6 and P2 1980, grow to high titre in MEK3 cells, and their structural proteins and virus-induced protein synthesis have been characterized by gel electrophoresis and immunoprecipitation. Purified viruses contain seven polypeptides, including cellular actin: L (175K mol. wt.), HN (72K to 74K), NP (66K to 67K), F1 (52K to 58K), P (49K), A (44.5K) and M (39K). Virus-induced polypeptide synthesis was first detected at 8 h post-infection with the appearance of NP; other major structural proteins were detected from 10 to 12 h after infection and onwards. The synthesis of both the structural glycoproteins was demonstrated, although proteolytic processing could not be detected. Reproducible differences in the gel migration of the HN, F1 and NP polypeptides were found in whole virus, in infected cells and cells subjected to immunoprecipitation. These differences may reflect genetic diversity within HPV2 and provide a means of probing the molecular epidemiology of these viruses.

AN 84009685 MEDLINE  
 DN 84009685 PubMed ID: 6311949  
 TI Characterization of human parainfluenza viruses. I. The structural proteins of parainfluenza virus 2 and their synthesis in infected cells.  
 AU Cowley J A; Barry R D  
 SO JOURNAL OF GENERAL VIROLOGY, (1983 Oct) 64 (Pt 10) 2117-25.  
 Journal code: I9B; 0077340. ISSN: 0022-1317.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 198311  
 ED Entered STN: 19900319  
 Last Updated on STN: 19970203  
 Entered Medline: 19831123

L5 ANSWER 9 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS  
 AB Type I interferon (IFN) induces antiviral responses through the activation of the ISGF3 transcription factor complex that contains the subunit proteins STAT1, STAT2, and p48/ISGF3gamma/IRF9. The ability of some human paramyxoviruses to overcome IFN actions by specific proteolysis of STAT proteins has been examined. Infection of cells with type 2, but not type 1 or type 3 human parainfluenza virus (HPIV) leads to a loss of cellular STAT2 protein. Expression of a single HPIV2 protein derived from the V open reading frame blocks IFN-dependent transcriptional responses in the absence of other viral proteins. The loss of IFN response is due to V-protein-induced proteolytic degradation of STAT2. Expression of HPIV2 V

causes the normally stable STAT2 protein to be rapidly degraded, and this proteolytic activity can be partially alleviated by proteasome inhibition.

No V-protein-specific effects on STAT2 mRNA levels were observed. The results indicate that the V protein of HPIV2 is sufficient to recognize and target a specific cellular transcription factor for destruction by cellular machinery.

AN 2001:280557 BIOSIS

DN PREV200100280557

TI The V protein of **human parainfluenza virus**

2 antagonizes type I interferon responses by destabilizing signal transducer and activator of transcription 2.

AU Parisien, Jean-Patrick; Lau, Joe F.; Rodriguez, Jason J.; Sullivan, Brian M.; Moscona, Anne; Parks, Griffith D.; Lamb, Robert A.; Horvath, Curt M.  
(1)

CS (1) Mount Sinai School of Medicine, One Gustave L. Levy Place, East Building Room 12-20D, New York, NY, 10029: curt.horvath@mssm.edu USA

SO Virology, (May 10, 2001) Vol. 283, No. 2, pp. 230-239. print.

ISSN: 0042-6822.

DT Article

LA English

SL English

L5 ANSWER 10 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

AB Acute respiratory diseases (ARD) are the most common infections in humans and difficult to prevent. Viruses have been recognized as predominant ethiological agents. In Cuba, ARD constitute a major problem of health

and are the first cause of morbidity and important cause of mortality. In this paper, rapid diagnosis was performed to 516 clinical samples which arrived

to the Reference Respiratory Viruses Laboratory of the Pedro Kouri Institute of Tropical Medicine (IPK) from different parts of Havana City during 1995, 1996 and 1997. The results obtained have shown 218 positive samples (Influenza A, 89; respiratory syncytial virus 52; Influenza B,

45; Adenovirus, 13; human parainfluenza virus (HPIV)-1, 6; HPIV-2, 3 and HPIV-3, 10). Influenza A was the virus most frequently found in adults, whereas in closed population of teen-agers and adults, Influenza B was frequently found. Furthermore, respiratory syncytial virus was the most important pathogen in childrens under 1 year of age.

AN 2000:214912 BIOSIS

DN PREV200000214912

TI Rapid diagnosis of the principal respiratory viruses by indirect immunofluorescence in Havana City.

AU Cancio, R. (1); Savon, C.; Oropeza, S.; Abreu, I.; Perez Martinez, T.; Hernandez, B.; Gonzalez, G.; Valdez, O.; Goyenechea, A.

CS (1) Departamento de Virologia, Instituto de Medicina Tropical Pedro Kouri (IPK), Marianao 13, Ciudad de la Habana Cuba

SO Revista Argentina de Microbiologia, (2000) Vol. 32, No. 1, pp. 21-26.

ISSN: 0325-7541.

DT Article

LA Spanish

SL English; Spanish

L5 ANSWER 11 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

AB To better define the contribution of human parainfluenza viruses (HPIVs) to lower respiratory tract infection in adults, we tested acute- and convalescent-phase serum specimens from hospitalized adults participating in a population-based prospective study of lower respiratory tract infection during 1991-1992. We tested all available specimens from the epidemic seasons for each virus and approx300 randomly selected specimens from the corresponding off-seasons for antibodies to HPIV-1, HPIV-2, or HPIV-3. During the respective epidemic season, HPIV-1 infection was

detected in 18 (2.5%) of 721 and HPIV-3 infection in 22 (3.1%) of 705 patients with lower respiratory tract infection. Only 2 (0.2%) of 1,057 patients tested positive for HPIV-2 infection. No HPIV-1 infections and only 2 (0.7% of 281 patients tested) HPIV-3 infections were detected during the off-seasons. HPIV-1 and HPIV-3 were among the four most frequently identified infections associated with lower respiratory tract infection during their respective outbreak seasons.

AN 1999:359452 BIOSIS

DN PREV199900359452

TI Parainfluenza virus infection among adults hospitalized for lower respiratory tract infection.

AU Marx, Arthur; Gary, Howard E., Jr.; Marston, Barbara J.; Erdman, Dean D.; Breiman, Robert F.; Torok, Thomas J.; Plouffe, Joseph F.; File, Thomas

M., Jr.; Anderson, Larry J. (1)

CS (1) Centers for Disease Control and Prevention, 1600 Clifton Road, Northeast, Atlanta, GA, 30333 USA

SO Clinical Infectious Diseases, (July, 1999) Vol. 29, No. 1, pp. 134-140. ISSN: 1058-4838.

DT Article

LA English

SL English

L5 ANSWER 12 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

AB The simian parainfluenza virus 5 (SV5) WP gene encodes two proteins; V and

the phosphoprotein P. The V and P proteins are amino coterminal for 164 residues, but they have unique carboxyl termini. The unique carboxyl terminus of V contains seven cysteine residues, resembles a zinc finger, and binds two atoms of zinc. In a glutathione-S-transferase (GST)-fusion protein selection of cell lysate assay, the GST-V protein was found to interact with the 127-kDa subunit (DDB1) of the damage-specific DNA binding protein (DDB) (also known as UV-damaged DNA binding protein (UV-DDB), xeroderma pigmentosum group E binding factor (XPE-BF), and the hepatitis B virus X-associated protein 1 (XAP-1)). A reciprocal GST-DDB1 fusion protein selection assay of SV5-infected cell lysates showed that DDB1 and V interact, and it was found that V and DDB1 could be coimmunoprecipitated from SV5-infected cells or from cells expressing V and DDB1 using the vaccinia virus T7 expression system. The interaction

of

V and DDB1 involves the carboxyl-terminal domain of V in that either deletion of the V carboxyl-terminal domain or substitution of the

cysteine

residues (C189, C193, C205, C207, C210, C214, and C217) in the zinc-binding domain with alanine was able to disrupt binding to DDB1. The V proteins of the mumps virus, **human parainfluenza virus 2** (hPIV2), and measles virus have also been found to interact with DDB1 in GST-fusion protein selection assays using in vitro transcribed and translated DDB1.

AN 1998:473690 BIOSIS

DN PREV199800473690

TI The V protein of the paramyxovirus SV5 interacts with damage-specific DNA binding protein.

AU Lin, Grace Y.; Paterson, Reay G.; Richardson, Christopher D.; Lamb, Robert

A.

CS Dep. Biochem. Mol. Biol. Cell Biol., 2153 North Campus Drive, Evanston, IL

60208-3500 USA

SO Virology, (Sept. 15, 1998) Vol. 249, No. 1, pp. 189-200. ISSN: 0042-6822.

DT Article

LA English

L5 ANSWER 13 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

AB A recombinant baculovirus expressing the nucleocapsid gene (NP) of Newcastle disease virus (NDV), a member of the genus Rubulavirus, has been generated and shown to express the native protein to high levels in insect cells. In contrast to the NP protein of the rubulavirus **human parainfluenza virus 2**, the NDV protein has been demonstrated by electron microscopy and caesium chloride gradient analysis to be capable of self-assembly in vivo to form nucleocapsid-like structures in the absence of other NDV proteins. These structures, which contained RNA that was resistant to micrococcal nuclease digestion, were also observed when the protein was expressed in *E. coli*, a phenomenon which was not inhibited by the presence of a 40 amino acid fusion region at the amino terminus of the protein. Further, the formation of these structures was inhibited by the co-expression of the phosphoprotein (P). Therefore, we conclude that the P protein acts as a chaperone, preventing uncontrolled encapsulation of non-viral RNA by NP protein.

AN 1997:433740 BIOSIS

DN PREV199799732943

TI Assembly of recombinant Newcastle disease virus nucleocapsid protein into nucleocapsid-like structures is inhibited by the phosphoprotein.

AU Errington, William; Emmerson, Peter T. (1)

CS (1) Dep. Biochem. Genetics, Med. Sch., Univ. Newcastle upon Tyne, Newcastle upon Tyne NE2 4HH UK

SO Journal of General Virology, (1997) Vol. 78, No. 9, pp. 2335-2339. ISSN: 0022-1317.

DT Article

LA English

L5 ANSWER 14 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

AB Antigenic relationships of simian virus 41 (SV41) to other paramyxoviruses

were examined by immunoprecipitation of isotope-labelled SV41-infected cell lysates with specific antisera. SV41 is closely related to the group comprising **human parainfluenza virus 2** (HPIV-2), simian virus 5 (SV5), parainfluenza virus 4 and mumps virus. Slight cross-neutralization was detected between SV41, HPIV-2 and SV5. Anti-SV41 activities were detected in 21 of 1116 human serum specimens, indicating that a proportion of the human population is infected with SV41. The haemagglutinin-neuraminidase of SV41 was preferentially immunoprecipitated by anti-SV41 positive sera.

AN 1990:518936 BIOSIS

DN BA90:136212

TI IMMUNOLOGICAL RELATIONSHIP OF SIMIAN VIRUS 41 SV41 TO OTHER PARAMYXOVIRUSES AND SEROLOGICAL EVIDENCE OF SV41 INFECTION IN HUMAN POPULATIONS.

AU NISHIO M; TSURUDOME M; BANDO H; ITO Y

CS DEP. MICROBIOL., MIE UNIV. SCH. MED., 2-174 EDOBASHI, TSU-SHI, MIE-PREFECTURE 514, JPN.

SO J GEN VIROL, (1990) 71 (9), 2093-2098.

CODEN: JGVIAY. ISSN: 0022-1317.

FS BA; OLD

LA English

L5 ANSWER 15 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

AB Two strains of **human parainfluenza virus**

**2** (HPV2), P2 1972/1976 and P2 1980, grow to high titer in [monkey embryo kidney] MEK3 cells, and their structural proteins and

virus-induced

protein synthesis have been characterized by gel electrophoresis and immunoprecipitation. Purified viruses contain 7 polypeptides, including cellular actin: L (175K MW), HN (72-74K), NP (66-67K), F1 (52-58K), P (489K), A (44.5K) and M (39K). Virus-induced polypeptide synthesis was first detected at 8 h postinfection with the appearance of NP; other

major



structural proteins were detected from 10 to 12 h after infection and onwards. The synthesis of both the structural glycoproteins was demonstrated, although proteolytic processing could not be detected. Reproducible differences in the gel migration of the HN, F1 and NP polypeptides were found in whole virus, in infected cells and cells subjected to immunoprecipitation. These differences may reflect genetic diversity within HPV2 and provide a means of probing the molecular epidemiology of these viruses.

AN 1984:215245 BIOSIS

DN BA77:48229

TI CHARACTERIZATION OF HUMAN PARAINFLUENZA VIRUSES 1. THE STRUCTURAL PROTEINS

OF PARAINFLUENZA VIRUS 2 AND THEIR SYNTHESIS IN INFECTED CELLS.

AU COWLEY J A; BARRY R D

CS FAC. MED., UNIV. NEWCASTLE, DAVID MADDISON CLIN. SCI. BUILD., ROYAL NEWCASTLE HOSP., NEWCASTLE, N.S.W. 2300, AUSTRALIA.

SO J GEN VIROL, (1983) 64 (10), 2117-2126.

CODEN: JGVIAY. ISSN: 0022-1317.

FS BA; OLD

LA English

=> d 12 1-20 ab bib

L2 ANSWER 1 OF 52 MEDLINE

AB In this study, we investigated the role of the membrane-proximal region of

the human parainfluenza virus type 2 (HPIV2) F protein by mutational analysis, including deletion, insertion, and substitution. Deletion or replacement of the entire 12 amino acid region (aa 474-485) of the HPIV2

F protein completely abolished its fusion activity when coexpressed with the

HPIV2 HN protein. Deletion of groups of four of aa 478-485, single alanine, or other amino acid substitutions among aa 478-485 had minimal

or limited effects on HPIV2 F/HN-induced cell fusion. However, a significant reduction in, or complete inhibition of, fusion activity was observed when

aa 474-477 were deleted, or the N475, F476, or F477 residues were singly substituted with alanine. In addition, insertions of four amino acids at this region or deletion of eight or more amino acids significantly

reduced F protein fusion activity. The oligomerization patterns and levels of cell

surface expression of the mutant F proteins were compared to those of the wild-type HPIV2 F protein. The mutant HPIV2 F proteins defective in

fusion were also found to be unable to initiate hemifusion, indicating that there

is a specific requirement for three specific amino acids as well as the spacing in this region for initiating lipid mixing. Copyright 2001 Academic Press.

AN 2001205389 MEDLINE

DN 21109121 PubMed ID: 11162818

TI Three membrane-proximal amino acids in the human parainfluenza type 2 (HPIV 2) F protein are critical for fusogenic activity.

AU Tong S; Yi F; Martin A; Yao Q; Li M; Compans R W

CS Department of Microbiology and Immunology, Emory University, Atlanta, Georgia 30322, USA.

NC CA 18611 (NCI)

SO VIROLOGY, (2001 Feb 1) 280 (1) 52-61.

Journal code: XEA; 0110674. ISSN: 0042-6822.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 200104  
ED Entered STN: 20010417  
Last Updated on STN: 20010417  
Entered PubMed: 20010222  
Entered Medline: 20010412

L2 ANSWER 2 OF 52 MEDLINE

AB Sequencing studies of limited regions of the human parainfluenza viruses (HPIVs) genomes have helped describe patterns of virus circulation and characterize institutional outbreaks of HPIVs-associated respiratory illness. In this study, we sequenced reverse transcription polymerase chain reaction (RT-PCR)-amplified HPIVs RNA obtained from a multiplex RT-PCR assay described previously for simultaneous detection of HPIV-1, 2 and 3. Differences in the nucleotide sequences of limited regions of the HN gene allowed us to distinguish temporally and geographically diverse HPIV isolates (43 HPIV-1, 7 HPIV-2, 12 HPIV-3 isolates from this and previously published studies). In addition, an outbreak of HPIV-3-associated illness among infants on a pediatric ward was investigated by comparing sequences of three ward isolates with three matched community controls. Sequences of all ward isolates were identical and differed from those of the community controls, suggesting a single introduction and nosocomial transmission of the virus. Combining

multiplex

reverse transcription polymerase chain reaction (RT-PCR) assays with direct sequencing of the PCR products can provide an integrated system

for

rapid diagnosis and characterization of HPIVs.

AN 2001021454 MEDLINE

DN 20377315 PubMed ID: 10921847

TI Rapid molecular epidemiologic studies of human parainfluenza viruses based

on direct sequencing of amplified DNA from a multiplex RT-PCR assay.

AU Echevarria J E; Erdman D D; Meissner H C; Anderson L

CS Division of Viral and Rickettsial Diseases, National Center for Infectious

Diseases, Centers for Disease Control and Prevention, Atlanta, GA, USA..  
jeecheva@isciii.es

SO JOURNAL OF VIROLOGICAL METHODS, (2000 Jul) 88 (1) 105-9.  
Journal code: HQR. ISSN: 0166-0934.

CY Netherlands

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

OS GENBANK-AF039922; GENBANK-AF039923; GENBANK-AF039924; GENBANK-AF039925

EM 200011

ED Entered STN: 20010322

Last Updated on STN: 20010322

Entered Medline: 20001109

L2 ANSWER 3 OF 52 MEDLINE

AB Eleven monoclonal antibodies (MAbs) directed against the large (L) protein

of human parainfluenza type 2 virus (hPIV-2) were prepared to examine the interactions of the L protein with other viral proteins. Coimmunoprecipitation assays using these MAbs revealed that the L protein directly interacted with the phospho- (P) and nucleocapsid (NP) proteins in vivo and in vitro. Mutational analysis of the P or NP protein was performed to identify the region(s) on these proteins interacting

with

L protein, indicating that amino acids 278-353 on the P protein and amino acids 403-494 on the NP protein are essential for the binding to the L protein.

AN 2000423529 MEDLINE  
DN 20377922 PubMed ID: 10915594  
TI Mapping of domains on the human parainfluenza type 2 virus P and NP proteins that are involved in the interaction with the L protein.  
AU Nishio M; Tsurudome M; Ito M; Ito Y  
CS Department of Microbiology, Mie University School of Medicine, 2-174, Edobashi, Tsu-Shi, Mie-Ken, 514-8507, Japan..  
nishio@doc.medic.mie-u.ac.jp  
SO VIROLOGY, (2000 Aug 1) 273 (2) 241-7.  
Journal code: XEA; 0110674. ISSN: 0042-6822.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 200009  
ED Entered STN: 20000915  
Last Updated on STN: 20000915  
Entered Medline: 20000906

L2 ANSWER 4 OF 52 MEDLINE  
AB Acute respiratory diseases (ARD) are the most common infections in humans and difficult to prevent. Viruses have been recognized as predominant ethiological agents. In Cuba, ARD constitute a major problem of health and are the first cause of morbidity and important cause of mortality. In this paper, rapid diagnosis was performed to 516 clinical samples which arrived to the Reference Respiratory Viruses Laboratory of the Pedro Kouri Institute of Tropical Medicine (IPK) from different parts of Havana City during 1995, 1996 and 1997. The results obtained have shown 218 positive samples (Influenza A, 89; respiratory syncytial virus 52; Influenza B, 45; Adenovirus, 13; human parainfluenza virus(HPIV)-1, 6; HPIV-2, 3 and HPIV-3, 10). Influenza A was the virus most frequently found in adults, whereas in closed population of teen-agers and adults, Influenza B was frequently found. Furthermore, respiratory syncytial virus was the most important pathogen in children's under 1 year of age.

AN 2000247663 MEDLINE  
DN 20247663 PubMed ID: 10785939  
TI [Rapid diagnosis of principal respiratory viruses in the city of Havana, 1995-97].  
Diagnostico rapido de los principales virus respiratorios en Ciudad de la Habana, 1995-97.  
AU Cancio R; Savon C; Oropeza S; Abreu I; Perez Martinez T; Hernandez B; Gonzalez G; Valdez O; Goyenechea A  
CS Departamento de Virologia, Instituto de Medicina Tropical Pedro Kouri (IPK), Ciudad de la Habana, Cuba.. rey@ipk.sld.cu  
SO REVISTA ARGENTINA DE MICROBIOLOGIA, (2000 Jan-Mar) 32 (1) 21-6.  
Journal code: QZ8; 8002834. ISSN: 0325-7541.  
CY Argentina  
DT Journal; Article; (JOURNAL ARTICLE)  
LA Spanish  
FS Priority Journals  
EM 200005  
ED Entered STN: 20000613  
Last Updated on STN: 20000613  
Entered Medline: 20000531

L2 ANSWER 5 OF 52 MEDLINE  
AB Two monoclonal antibodies (mAbs) specific for the human parainfluenza virus type 2 (hPIV-2) V protein were obtained by immunizing mice with the V protein recombinantly expressed in Escherichia

coli. Both mAbs were found to react with the V protein in ELISA and in Western blot analysis. Using these mAbs and previously obtained mAbs specific for hPIV-2 nucleoprotein (NP) or hPIV-2 phospho-(P) protein, we examined the intracellular distributions of the V, P and NP proteins in hPIV-2-infected cells by indirect immunofluorescence analyses. The P and NP proteins were organized in numerous granules in the cytoplasm of hPIV-2 infected cells. In contrast, the V protein showed diffuse nuclear and cytoplasm distributions.

AN 2000214733 MEDLINE  
 DN 20214733 PubMed ID: 10753059  
 TI Isolation of monoclonal antibodies directed against the V protein of human

parainfluenza virus type 2 and localization of the V protein in virus-infected cells.

AU Nishio M; Tsurudome M; Ito M; Kawano M; Kusagawa S; Komada H; Ito Y  
 CS Department of Microbiology, Mie University School of Medicine, Japan.. nishio@doc.medic.mie-u.ac.jp  
 SO MEDICAL MICROBIOLOGY AND IMMUNOLOGY, (1999 Nov) 188 (2) 79-82.  
 Journal code: M58; 0314524. ISSN: 0300-8584.  
 CY GERMANY: Germany, Federal Republic of  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 200005  
 ED Entered STN: 20000512  
 Last Updated on STN: 20000512  
 Entered Medline: 20000502

L2 ANSWER 6 OF 52 MEDLINE

AB We describe a multiplex reverse transcription-PCR (m-RT-PCR) assay that is

able to detect and differentiate all known human parainfluenza viruses (HPIVs). Serial dilution experiments with reference strains that compared cell culture isolation and m-RT-PCR showed sensitivities ranging from 0.0004 50% tissue culture infective dose (TCID<sub>50</sub>) for HPIV type 4B (HPIV-4B) to 32 TCID<sub>50</sub>s for HPIV-3. As few as 10 plasmids containing HPIV PCR products could be detected in all cases. When 201 nasopharyngeal aspirate specimens from pediatric patients hospitalized for lower respiratory illness were tested, m-RT-PCR assay detected 64 HPIVs (24 HPIV-3, 23 HPIV-1, 10 HPIV-4, and 7 HPIV-2), while only 42 of them (21 HPIV-1, 14 HPIV-3, 6 HPIV-2, and 1 HPIV-4 isolates) grew in cell culture. Our m-RT-PCR assay was more sensitive than either cell culture isolation or indirect immunofluorescence with monoclonal antibodies for the detection of HPIV infections. Also, HPIV-4 was more frequently detected than HPIV-2 in this study, suggesting that it may have been underestimated as a lower respiratory tract pathogen because of the insensitivity of

cell culture.

AN 2000164696 MEDLINE  
 DN 20164696 PubMed ID: 10699020  
 TI Detection and identification of human parainfluenza viruses 1, 2, 3, and 4

in clinical samples of pediatric patients by multiplex reverse transcription-PCR.

CM Erratum in: J Clin Microbiol 2000 Jul;38(7):2805  
 AU Aguilar J C; Perez-Brena M P; Garcia M L; Cruz N; Erdman D D; Echevarria J  
 E  
 CS Servicio de Virologia, Centro Nacional de Microbiologia, Instituto de Salud Carlos III, Carretera de Majadahonda Pozuelo s/n, 28220 Majadahonda, Madrid, Spain.. jaguilar@isciii.es  
 SO JOURNAL OF CLINICAL MICROBIOLOGY, (2000 Mar) 38 (3) 1191-5.

Journal code: HSH; 7505564. ISSN: 0095-1137.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 200004  
ED Entered STN: 20000413  
Last Updated on STN: 20001027  
Entered Medline: 20000405

L2 ANSWER 7 OF 52 MEDLINE  
AB The epitopes recognized by 41 monoclonal antibodies directed against the nucleocapsid protein (NP) of human parainfluenza virus type 2 (hPIV-2) were mapped on the primary structure of the hPIV-2 NP protein by testing their reactivities with deletion mutants. By Western immunoblotting using these monoclonal antibodies, the analysis of deletion mutants of the hPIV-2 NP protein was performed to identify the region essential for NP-NP interaction and phosphoprotein (P)-binding sites on the NP protein. The results indicate that the N-terminal 294 aa of the NP protein are all required for NP-NP self-assembly, and that two C-terminal parts of the NP protein are essential for NP-P binding: one region, aa 295-402, is required for binding to the C-terminal part of the P protein and another region, aa 403-494, to the N-terminal part of the P protein.

AN 1999394675 MEDLINE  
DN 99394675 PubMed ID: 10466799  
TI Mapping of domains on the human parainfluenza virus type 2 nucleocapsid protein (NP) required for NP-phosphoprotein or NP-NP interaction.  
AU Nishio M; Tsurudome M; Ito M; Kawano M; Kusagawa S; Komada H; Ito Y  
CS Department of Microbiology, Mie University School of Medicine, Tsu-Shi, Mie-Ken, Japan.. nishio@doc.medic.mie-u.ac.jp  
SO JOURNAL OF GENERAL VIROLOGY, (1999 Aug) 80 ( Pt 8) 2017-22.  
Journal code: I9B; 0077340. ISSN: 0022-1317.

CY ENGLAND: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 199909  
ED Entered STN: 19991012  
Last Updated on STN: 19991012  
Entered Medline: 19990927

L2 ANSWER 8 OF 52 MEDLINE  
AB The antiviral activity of Sanicula europaea L. extracts against human parainfluenza virus type 2 (HPIV-2) was examined. The extract prepared from the leaves of the plant and a fraction separated from the crude extract with gel filtration chromatography were found to inhibit HPIV-2 replication without any toxic effect on Vero cells. The acidic fraction obtained from the crude extract of S. europaea leaves was found to be the most active fraction with plaque inhibition assay at non-cytotoxic concentrations. Unfortunately, antiviral activity was not detected in the molecules purified from the crude ethanol extract of Sanicula leaves.  
Copyright 1999 John Wiley & Sons, Ltd.

AN 1999373528 MEDLINE  
DN 99373528 PubMed ID: 10441789  
TI Antiviral activity of Sanicula europaea L. extracts on multiplication of human parainfluenza virus type 2.  
AU Karagoz A; Arda N; Goren N; Nagata K; Kuru A  
CS Molecular Biology Section, Department of Biology, Faculty of Science, University of Istanbul, Vezneciler, 34459-Istanbul, Turkey.  
SO PHYTOTHERAPY RESEARCH, (1999 Aug) 13 (5) 436-8.  
Journal code: C6Y; 8904486. ISSN: 0951-418X.

CY ENGLAND: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 199910  
ED Entered STN: 19991014  
Last Updated on STN: 19991014  
Entered Medline: 19991007

L2 ANSWER 9 OF 52 MEDLINE

AB To better define the contribution of human parainfluenza viruses (HPIVs) to lower respiratory tract infection in adults, we tested acute- and convalescent-phase serum specimens from hospitalized adults participating in a population-based prospective study of lower respiratory tract infection during 1991-1992. We tested all available specimens from the epidemic seasons for each virus and approximately 300 randomly selected specimens from the corresponding off-seasons for antibodies to HPIV-1, HPIV-2, or HPIV-3. During the respective epidemic season, HPIV-1 infection was detected in 18 (2.5%) of 721 and HPIV-3 infection in 22 (3.1%) of 705 patients with lower respiratory tract infection. Only 2 (0.2%) of 1,057 patients tested positive for HPIV-2 infection. No HPIV-1 infections and only 2 (0.7% of 281 patients tested) HPIV-3 infections were detected during the off-seasons. HPIV-1 and HPIV-3 were among the four most frequently identified infections associated with lower respiratory tract infection during their respective outbreak seasons.

AN 1999360958 MEDLINE

DN 99360958 PubMed ID: 10433576

TI Parainfluenza virus infection among adults hospitalized for lower respiratory tract infection.

AU Marx A; Gary H E Jr; Marston B J; Erdman D D; Breiman R F; Torok T J; Plouffe J F; File T M Jr; Anderson L J

CS Division of Viral and Rickettsial Diseases, National Center for Infectious

Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia 30333, USA.

SO CLINICAL INFECTIOUS DISEASES, (1999 Jul) 29 (1) 134-40.

Journal code: A4J; 9203213. ISSN: 1058-4838.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199909

ED Entered STN: 19990925

Last Updated on STN: 19990925

Entered Medline: 19990916

L2 ANSWER 10 OF 52 MEDLINE

AB We reviewed immunological relationships between paramyxoviruses and molecular evolution of paramyxoviruses. (1) paramyxoviruses are divided into two groups by the basis of the immunological relationship, i.e., parainfluenza type 1 virus group and type 2 virus group; (2) according to the NP and M proteins sequences, paramyxoviruses are divided into parainfluenza type 1 virus and type 2 virus groups; the former group is composed of HPIV-1, SV, HPIV-3 and BPIV-3, and the latter group consists of HPIV-2, SV41, SV5, MuV, HPIV-4A and HPIV-4B; and this grouping coincides with that by immunological relationships; (3) alignment of the P and V proteins of paramyxoviruses demonstrates that irregular gaps are present around the RNA-editing sites, suggesting that dynamic processes have been occurred during molecular evolution; (4) the NP proteins of the morbilliviruses are more related to the parainfluenza type 2 group, while the M proteins show closer-relationship with the parainfluenza type 1 group; (5) the parainfluenza type 2 group can be subdivided into two groups, i.e., mammal and avian types; (6) phylogenetic trees for mononegaviruses are constructed.

AN 97446844 MEDLINE  
DN 97446844 PubMed ID: 9301318  
TI Molecular evolution of paramyxoviruses.  
AU Ito Y  
CS Department of Microbiology, Mie University School of Medicine.  
SO NIPPON RINSHO. JAPANESE JOURNAL OF CLINICAL MEDICINE, (1997 Sep) 55 (9)  
2476-83. Ref: 20  
Journal code: KIM; 0420546. ISSN: 0047-1852.  
CY Japan  
DT Journal; Article; (JOURNAL ARTICLE)  
General Review; (REVIEW)  
(REVIEW LITERATURE)  
LA Japanese  
FS Priority Journals  
EM 199712  
ED Entered STN: 19980109  
Last Updated on STN: 19980109  
Entered Medline: 19971216

L2 ANSWER 11 OF 52 MEDLINE  
AB The epitopes recognized by 42 monoclonal antibodies directed against the human parainfluenza virus type 2 (hPIV-2) phosphoprotein (P) were mapped on the primary structure of the P protein by testing their reactivities with deletion mutants. By Western Immunoblotting with these monoclonal antibodies and P protein deletion mutants the region essential for P-P interactions was determined. The P protein region encompassing amino acids 211-248 was required for proper folding and oligomerization which is mediated by predicted coiled-coils

in this region. The oligomer was shown to be a homotrimer by chemical cross-linking experiments.

AN 97335256 MEDLINE  
DN 97335256 PubMed ID: 9191922  
TI Human parainfluenza virus type 2 phosphoprotein: mapping of monoclonal antibody epitopes and location of the multimerization domain.  
AU Nishio M; Tsurudome M; Ito M; Watanabe N; Kawano M; Komada H; Ito Y  
CS Department of Microbiology, Mie University School of Medicine, Japan..  
nishio@doc.medic.mie-u.ac.jp  
SO JOURNAL OF GENERAL VIROLOGY, (1997 Jun) 78 ( Pt 6) 1303-8.  
Journal code: I9B; 0077340. ISSN: 0022-1317.  
CY ENGLAND: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 199707  
ED Entered STN: 19970721  
Last Updated on STN: 19970721  
Entered Medline: 19970703

L2 ANSWER 12 OF 52 MEDLINE  
AB Syncytium formation and subsequent generalized cell fusion have been reported as potentially important mechanisms of virus-induced cytotoxic effects. We tried to clarify the roles of fusion regulatory factor-1 (FRP-1) in virus-induced cell fusion. Two mutated human FRP-1/CD98 proteins [FRP-1/HN, in which the cytoplasmic domain was replaced with the cytoplasmic domain of human parainfluenza virus type 2 (HPIV-2) haemagglutinin-neuraminidase (HN), and FRP-1/330 (serine), in which a cysteine at amino acid 330 was mutated to serine], when expressed stably in L929 cells, were lacking in cell-fusion-enhancing activity stimulated by anti-FRP-1 antibodies. Anti-FRP-1 antibodies enhanced Newcastle disease virus (NDV)-mediated polykaryocyte formation in parent HeLa cells, while anti-FRP-1 antibodies showed no/low effect on polykaryocyte formation in NDV-infected HeLa cells constitutively expressing FRP-1/HN (HeLa-FRP-1/HN cells), indicating that the FRP-1/HN molecule is capable of acting as a dominant negative inhibitor.

Furthermore, when HeLa-FRP-1/HN cells were infected with various rubulaviruses (HPIV-2, mumps virus, simian viruses 5 and 41), virus-induced cell fusion was also suppressed, although virus replication was not inhibited in these cells, showing that FRP-1 molecules are required for virus-induced cell fusion. Therefore, FRP-1 is considered to be related to the pathogenesis of paramyxoviruses.

AN 97275888 MEDLINE  
 DN 97275888 PubMed ID: 9129649  
 TI Paramyxovirus-induced syncytium cell formation is suppressed by a dominant negative fusion regulatory protein-1 (FRP-1)/CD98 mutated construct: an important role of FRP-1 in virus-induced cell fusion.  
 AU Okamoto K; Ohgimoto S; Nishio M; Tsurudome M; Kawano M; Komada H; Ito M; Sakakura Y; Ito Y  
 CS Department of Microbiology, Mie University School of Medicine, Japan.  
 SO JOURNAL OF GENERAL VIROLOGY, (1997 Apr) 78 ( Pt 4) 775-83.  
 Journal code: I9B; 0077340. ISSN: 0022-1317.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199705  
 ED Entered STN: 19970609  
 Last Updated on STN: 19970609  
 Entered Medline: 19970523

L2 ANSWER 13 OF 52 MEDLINE  
 AB Fusion regulatory protein-1 (FRP-1) regulates virus-mediated cell fusion and induces poly-karyocyte formation of monocytes without any fusogen. We have recently reported that FRP-1 and the 4F2/CD98 heavy chain are identical molecules. Cell fusion in Newcastle disease virus (NDV)-infected HeLa cells was enhanced when cells were incubated with anti-FRP-1 MAb. Anti-FRP-1 MAbs also induced human immunodeficiency virus gp160-mediated cell fusion. However, HBJ127, an anti-FRP-1/4F2/CD98 MAb that enhanced cell fusion in NDV-infected cells, delayed human parainfluenza virus type 2 (HPIV-2)-induced cell fusion in HeLa cells, although these viruses belong to the same genus Rubulavirus. No anti-FRP-1 MAbs enhanced cell fusion in HPIV-2-infected HeLa cells. Anti-FRP-1 MAbs including HBJ127 showed no effect on virus growth and expression levels of virus-specific poly-peptides in HPIV-2-infected HeLa cells, indicating that the delay in cell fusion by an anti-FRP-1 MAb is not due to suppression of virus replication. When HeLa cells were transfected with an expression vector harbouring HPIV-2 HN and F genes, cell fusion was also suppressed by HBJ127, but the effect was weak in comparison with virus-infected cells. These data indicate anti-FRP-1 antibodies not only induce/enhance, but also inhibit/delay virus-induced cell fusion and therefore FRP-1 molecules are multifunctional.

AN 97163469 MEDLINE  
 DN 97163469 PubMed ID: 9010289  
 TI An anti-fusion regulatory protein-1 monoclonal antibody suppresses human parainfluenza virus type 2-induced cell fusion.  
 AU Okamoto K; Tsurudome M; Ohgimoto S; Kawano M; Nishio M; Komada H; Ito M; Sakakura Y; Ito Y  
 CS Department of Microbiology, Mie University School of Medicine, Edobashi, Tsu-Shi, Japan.  
 SO JOURNAL OF GENERAL VIROLOGY, (1997 Jan) 78 ( Pt 1) 83-9.  
 Journal code: I9B; 0077340. ISSN: 0022-1317.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals



EM 199702  
ED Entered STN: 19970306  
Last Updated on STN: 19970306  
Entered Medline: 19970224

L2 ANSWER 14 OF 52 MEDLINE

AB The paramyxovirus phospho- (P) and nucleocapsid (NP) proteins are involved in transcription and replication of the viral genome. To study the interaction between NP and P proteins, we established HeLa cell lines that constitutively expressed the NP and/or P proteins of human parainfluenza virus type 2 (hPIV-2). Co-immunoprecipitation assays revealed that the NP and P proteins can form complexes in HeLa cells expressing both proteins (HeLa-NP+P cells) and in mixed cell lysates of HeLa-NP and HeLa-P cells. Deletion mutant analysis of the P protein was performed to identify the regions of P protein that interact with NP protein. The results indicate that two independent NP-binding sites exist on P protein: one is located in the N-terminal part of the protein, aa 1-47, and the other in the C-terminal part, aa 357-395. In addition, cells co-expressing NP and P proteins with N-terminal deletions showed immunofluorescence staining patterns (granular pattern) similar to those found in hPIV-2-infected cells. However, cells co-expressing NP and P proteins with C-terminal deletions showed a different immunofluorescence staining pattern (diffuse pattern), indicating that the C-terminal region is required for granule formation.

AN 97042278 MEDLINE

DN 97042278 PubMed ID: 8887478

TI Interaction between nucleocapsid protein (NP) and phosphoprotein (P) of human parainfluenza virus type 2: one of the two NP binding sites on P is essential for granule formation.

AU Nishio M; Tsurudome M; Kawano M; Watanabe N; Ohgimoto S; Ito M; Komada H; Ito Y

CS Department of Microbiology, Mie University School of Medicine, Tsu-Shi, Japan.. nishio@doc.medic.mie-u.ac.jp

SO JOURNAL OF GENERAL VIROLOGY, (1996 Oct) 77 ( Pt 10) 2457-63.  
Journal code: I9B; 0077340. ISSN: 0022-1317.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199612

ED Entered STN: 19970128  
Last Updated on STN: 19980206  
Entered Medline: 19961213

L2 ANSWER 15 OF 52 MEDLINE

AB Interaction of the nucleocapsid (NP) and V proteins of human parainfluenza type 2 virus (hPIV-2) was investigated using a transient expression system. When the NP proteins were co-expressed with the V proteins, some of the NP proteins were translocated into the nuclei.

These findings suggest that the NP protein interact with the V proteins. We examined the interaction of the NP proteins and the P, V proteins or deletion mutants of V protein using immunofluorescence and co-immunoprecipitation plus Western blotting analyses, and showed that

the

V proteins of hPIV-2 bind to the NP proteins and that the N-terminal domain of V protein interacts directly with the NP proteins. When the NP proteins were co-expressed with the V proteins or the N-terminal fragments (aa 1-46), the NP proteins were detected diffusely in the nuclei of the transfected cells, and were also detected in cytoplasmic inclusions. The NP and V proteins were co-localized in the

nuclei or cytoplasm. Furthermore, the NP proteins were co-precipitated with the P, V, and V (1-164) proteins by a specific antibody. The P proteins interact more closely with the NP proteins than do the V proteins. These findings indicate that the V proteins have the ability to bind the NP proteins.

AN 97039159 MEDLINE  
DN 97039159 PubMed ID: 8884740  
TI Binding of the V proteins to the nucleocapsid proteins of human parainfluenza type 2 virus.  
AU Watanabe N; Kawano M; Tsurudome M; Nishio M; Ito M; Ohgimoto S; Suga S; Komada H; Ito Y  
CS Department of Microbiology, Mie University School of Medicine, Japan.  
SO MEDICAL MICROBIOLOGY AND IMMUNOLOGY, (1996 Sep) 185 (2) 89-94.  
Journal code: M58; 0314524. ISSN: 0300-8584.  
CY GERMANY: Germany, Federal Republic of  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 199702  
ED Entered STN: 19970305  
Last Updated on STN: 19970305  
Entered Medline: 19970218

L2 ANSWER 16 OF 52 MEDLINE

AB The detection and quantitation of human parainfluenza virus type 1 (HPIV-1) RNA in nasal wash specimens from 49 children with lower respiratory infections were performed by a reverse transcription-PCR-enzyme hybridization assay (RT-PCR-EHA). The HPIV-1 RT-PCR-EHA was then used to test 40 samples from asymptomatic children. Primers and probes were designed from regions within the HPIV-1 hemagglutinin-neuraminidase gene which are highly conserved among all known genotypes. HPIV-1 was detected in all nine children who were culture positive. Other common respiratory viruses (HPIV-2, -3, and -4, mumps virus, respiratory syncytial virus, and influenza virus) were not detected by

the

HPIV-1 assay. Forty symptomatic children were negative by culture, and four of these were positive by RT-PCR-EHA. All of the samples from asymptomatic children were negative by culture and RT-PCR-EHA. RT-PCR-EHA was 100% sensitive (95% confidence interval, 0.66 to 1.00) and 95% specific (95% confidence interval, 0.88 to 0.99) compared with culture. The four false-positive results (relative to the results of culture) were in children with lower respiratory infections compatible with HPIV-1 infection and suggest that RT-PCR-EHA may be more sensitive than culture. Our data indicate that HPIV-1 may be underdiagnosed by routine culturing methods. RT-PCR-EHA has been demonstrated to be an easy, rapid, sensitive, and specific test for diagnosing HPIV-1 infection and provides a methodology for the rapid detection of closely related respiratory viruses.

AN 96415973 MEDLINE  
DN 96415973 PubMed ID: 8818880  
TI Rapid diagnosis of human parainfluenza virus type 1 infection by quantitative reverse transcription-PCR-enzyme hybridization assay.  
AU Fan J; Henrickson K J  
CS Department of Pediatrics, Medical College of Wisconsin, Milwaukee 53226, USA.  
SO JOURNAL OF CLINICAL MICROBIOLOGY, (1996 Aug) 34 (8) 1914-7.  
Journal code: HSH; 7505564. ISSN: 0095-1137.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 199612  
ED Entered STN: 19970128  
Last Updated on STN: 19980206

L2 ANSWER 17 OF 52 MEDLINE

AB In human parainfluenza virus type 2 (hPIV-2)-infected cells, anti-phosphoprotein (P)-specific monoclonal antibody (MAb) densely stained the perinuclear regions of infected cells throughout infection, indicating that the P protein was localized exclusively in the cell cytoplasm. By contrast, antigens recognized by MAbs directed against the P-V-common domain of hPIV-2 were located predominantly in the cytoplasm, but in some hPIV-2-infected cells they were also found in the nuclei, suggesting that a fraction of hPIV-2 V protein is localized there. hPIV-2 V protein expressed from a cDNA clone was localized in the nuclei of transfected cells. By using indirect immunofluorescence analyses, we examined the intracellular localization of various sequentially deleted V proteins, to determine the nuclear localization signals (NLS) of the V protein. Two noncontiguous regions in the V protein

were required for nuclear localization and retention, since deletion of these regions [region I (aa 1-46) and region II (aa 175-196)] resulted in cytoplasmic localization. Both regions resulted in nuclear localization independently. A nucleoplasmin-like NLS was identified in region II but

no consensus targeting sequence could be found in region I. When NP protein was co-expressed with V protein or the N-terminal fragment (aa 1-46) of V protein, a fraction of the NP protein was translocated into cell nuclei.

AN 96226024 MEDLINE

DN 96226024 PubMed ID: 8627237

TI Identification of the sequences responsible for nuclear targeting of the V

protein of human parainfluenza virus type 2.

AU Watanabe N; Kawano M; Tsurudome M; Kusagawa S; Nishio M; Komada H; Shima T; Ito Y

CS Department of Microbiology, Mie University School of Medicine, Japan.

SO JOURNAL OF GENERAL VIROLOGY, (1996 Feb) 77 ( Pt 2 ) 327-38.

Journal code: I9B; 0077340. ISSN: 0022-1317.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199606

ED Entered STN: 19960708

Last Updated on STN: 19960708

Entered Medline: 19960627

L2 ANSWER 18 OF 52 MEDLINE

AB cDNAs encoding human parainfluenza virus type 4A and type 4B (hPIV-4A and -4B) fusion (F) proteins were cloned and sequenced. The predicted amino acid sequences of the F proteins had similar characteristic traits to those reported for the F proteins of other paramyxoviruses. They were

more closely related to the F proteins of simian virus 5 (SV5), mumps virus (MuV), hPIV-2 and Newcastle disease virus (NDV) than to the F proteins of hPIV-1, hPIV-3, Sendai virus (SV) and measles virus (MV). In addition, hPIV-4A, hPIV-4B, SV5 and MuV shared a common feature of genomic organization: there was a small ORF between the F and haemagglutinin-neuraminidase (HN)-coding sequences, implying a common ancestry.

AN 96112211 MEDLINE

DN 96112211 PubMed ID: 8847531

TI Sequence analyses of human parainfluenza virus type 4A and type 4B fusion proteins.

AU Komada H; Bando H; Ito M; Ohta H; Kawano M; Nishio M; Tsurudome M; Watanabe N; Ikemura N; Kusagawa S; +

CS Department of Microbiology, Mie University School of Medicine, Japan.

SO JOURNAL OF GENERAL VIROLOGY, (1995 Dec) 76 ( Pt 12) 3205-10.  
Journal code: I9B; 0077340. ISSN: 0022-1317.  
CY ENGLAND: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
OS GENBANK-D49821; GENBANK-D49822  
EM 199610  
ED Entered STN: 19961106  
Last Updated on STN: 19961106  
Entered Medline: 19961021

L2 ANSWER 19 OF 52 MEDLINE

AB The RNA species synthesized in HPIV-2 infected CV-1 cells were identified and characterized. The largest RNA of approximately  $5.5 \times 10^6$  in molecular weight (MW) based on electrophoretic mobility, was identified as the genomic RNA. The other small RNA species of MWs  $2.4 \times 10^6$ ,  $1.1 \times 10^6$ ,  $0.77 \times 10^6$ ,  $0.68 \times 10^6$  and  $0.5 \times 10^6$  were identified as mRNAs. The five smallest RNAs were also synthesized in

vitro

and comigrated with RNAs synthesized in virus-infected cells. mRNAs synthesized both in vitro and in virus-infected cells were translated in vitro. NP, P, M and V proteins synthesized in vitro comigrated, when analyzed by SDS-PAGE, with the authentic proteins synthesized in virus-infected cells. Additionally, peptide mapping showed that the NP, P and M proteins synthesized in vitro were indistinguishable from their counterparts synthesized in infected cells. Analysis of the proteins from virions identified L, HN, NP, F (F1, F2), P, M and V proteins as virion structural proteins. Electrophoretic mobility of reduced and nonreduced F proteins was found to be different due to the conformational changes conferred by disulfide bonds.

AN 95282503 MEDLINE

DN 95282503 PubMed ID: 7762291

TI Characterization of human parainfluenza virus type 2 RNAs in infected cells and by in vitro synthesis.

AU Huang Y T; Romito R R; Panin M

CS Institute of Pathology, Case Western Reserve University, Cleveland, Ohio 44106-4907, USA.

SO VIRUS RESEARCH, (1995 Feb) 35 (2) 181-92.

Journal code: X98; 8410979. ISSN: 0168-1702.

CY Netherlands

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199506

ED Entered STN: 19950707

Last Updated on STN: 19950707

Entered Medline: 19950626

L2 ANSWER 20 OF 52 MEDLINE

AB To determine the morbidity, costs, and epidemiological features of lower respiratory tract infections (LRIs) due to human parainfluenza virus types

1 and 2 (HPIV-1 and HPIV-2), we evaluated 1,213 children < 6 years of age who were seen for LRIs in the emergency room of the Children's Hospital of Wisconsin and/or were admitted to the hospital for LRIs during the fall quarter of 1991. The age, sex, race, and respiratory syndrome were recorded for each child; 158 patients (13%) had respiratory samples cultured for viruses and were followed clinically for the duration of their illness. Caucasian children had croup diagnosed

more

often than did African-American children (relative risk [RR] = 3.12; 95% confidence interval [CI], 2.43-4.00;  $P < .001$ ), while African-American children more often had pneumonia (RR = 1.85; 95% CI, 1.36-2.5;  $P <$

.001).

Forty-five of 70 viruses recovered were HPIV-1 (17 cases) or HPIV-2 (28 cases). Together these two viruses were recovered from 49% of children presenting with croup, 10% of those presenting with bronchiolitis, and 12% of those presenting with pneumonia. Gender- and race-associated differences were documented in the group of children infected with HPIV-2: specifically, this group included more girls than boys (RR = 1.99; 95% CI, 1.02-3.88; P < .04) and more Caucasian than African-American children (RR = 2.64; 95% CI, 1.05-6.63; P = .027). These data extrapolate nationally to approximately 250,000 emergency-room visits and approximately 70,000 hospitalizations due to HPIV-1 and HPIV-2, with a cost of \$50 million for the former and \$140 million for the latter.

AN 94355511 MEDLINE  
DN 94355511 PubMed ID: 8075269  
TI Epidemiology and cost of infection with human parainfluenza virus types 1 and 2 in young children.  
AU Henrickson K J; Kuhn S M; Savatski L L  
CS Department of Pediatrics, Medical College of Wisconsin, Milwaukee.  
NC AI-31030 (NIAID)  
SO CLINICAL INFECTIOUS DISEASES, (1994 May) 18 (5) 770-9.  
Journal code: A4J; 9203213. ISSN: 1058-4838.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
(MULTICENTER STUDY)  
LA English  
FS Priority Journals  
EM 199410  
ED Entered STN: 19941013  
Last Updated on STN: 19941013  
Entered Medline: 19941003